Eduard Guasch, MD
Begoña Benito, MD
***Stanley Nattel, MD**

*Montreal Heart Institute/
Université de Montréal Research Center
5000 Belanger Street East
Montreal, Quebec H1T 1C8
Canada
E-mail: stanley.nattel@icm-mhi.org

http://dx.doi.org/10.1016/j.jacc.2013.07.102

Please note: Drs. Guasch and Benito contributed equally to this work.

REFERENCES

1. Guasch E, Benito B, Qi XY, et al. Atrial fibrillation promotion by endurance exercise: demonstration and mechanistic exploration in an animal model. J Am Coll Cardiol 2013;62:68–77.
2. Gay-Jordi G, Guash E, Benito B, et al. Losartan prevents heart fibrosis induced by long-term intensive exercise in an animal model. PLoS ONE 2013;8:e55427.
3. La Gerche A, Heidbuchel H, Burns AT, et al. Disproportionate exercise load and remodeling of the athlete's right ventricle. Med Sci Sports Exerc 2011;43:974–81.
4. La Gerche A, Burns AT, Mooney DJ, et al. Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes. Eur Heart J 2012;33:998–1006.
5. Burstein B, Libby E, Calderone A, Nattel S. Differential behaviors of atrial versus ventricular fibroblasts: a potential role for platelet-derived growth factor in atrial-ventricular remodeling differences. Circulation 2008;117:1630–41.
6. Mont L, Tamborero D, Elosua R, et al. Physical activity, height, and left atrial size are independent risk factors for lone atrial fibrillation in middle-aged healthy individuals. Europace 2008;10:15–20.

The Challenge of Voltage Channels

We read with great interest the report by Mountantonakis et al. (1). In this study, the authors reported that ventricular tachycardia (VT) isthmus sites were contained within channels in only 37% of voltage channels, and raised concerns about the suitability of using such channels as a target for ablation of unmappable VT. The authors came to this conclusion after analyzing a subgroup of 24 patients from a group of 140 patients who underwent VT ablation. These findings differ from previous data in which the majority of channels were related with clinical or inducible VTs (2,3).

Could evidence derived from 17% of patients with monomorphic VT be applied to the great majority of VT patients? In our opinion, great care should be exercised in extrapolating these results to the full spectrum of VT because of these points:

1. Complete activation mapping during VT was not obtained, and the circuit exit sites were not identified. Therefore, a connection between the isthmus site and a close channel cannot be completely excluded.
2. VT isthmuses not related to conduction channels are shown in maps in which the lower voltage limit is set at 0.5 mV. Could it be that isthmus sites were in incomplete channels connected to the main channel, as it seems to occur in Figure 5A? In previous studies, VT-related channels were always located within dense scar defined by a voltage <0.5 mV (2).
3. Voltage channels were identified during sinus rhythm and VT isthmus sites during VT. How these sites were later incorporated into voltage maps is very relevant, taking into account that ventricular volume and spatial position of the left ventricle may change during VT.
4. The average cycle length of channel-related VT was significantly shorter than that of VT not related to channels (377 ± 67 ms vs. 440 ± 40 ms, p = 0.01), but similar to the VT cycle length (374 ± 59 ms) reported in the previous study in which the majority of VT isthmuses were in channels (2). Could it be that fast VT isthmuses are commonly located in voltage channels? Slow VT may have a more complex substrate in which differentiation of central isthmuses from the surrounding scar could be more challenging. If slow mappable VTs have a different substrate, studies based on entrainment mapping could introduce bias when studying the relationship of VT isthmuses and voltage channels.

Pablo Ávila, MD
***Ángel Arenal, MD, PhD**

*Electrophysiology Unit
Cardiology Department
Hospital General Universitario Gregorio Marañón
46 Doctor Esquerdo Street
28007 Madrid
Spain
E-mail: arenal@secardiologia.es

http://dx.doi.org/10.1016/j.jacc.2013.06.067

REFERENCES

1. Mountantonakis SE, Park RE, Frankel DS, et al. Relationship between voltage map "channels" and the location of critical isthmus sites in patients with post-infarction cardiomyopathy and ventricular tachycardia. J Am Coll Cardiol 2013;61:2088–95.
2. Arenal A, del Castillo S, Gonzalez-Torrecilla E, et al. Tachycardia-related channel in the scar tissue in patients with sustained monomorphic ventricular tachycardias: influence of the voltage scar definition. Circulation 2004;110:2568–74.
3. Hsia HH, Lin D, Sauer WH, Callans DJ, Marchlinski FE. Anatomic characterization of endocardial substrate for hemodynamically stable reentrant ventricular tachycardia: identification of endocardial conducting channels. Heart Rhythm 2006;3:503–12.

Reply

The Challenge of Voltage Channels

We would like to thank Drs. Ávila and Arenal for their comments and interest in our work (1). The demonstration of voltage "channels" during ablation of ventricular tachycardia (VT) was originally proposed in the seminal studies by Arenal et al. (2) and Hsia et al. (3); we do believe this finding continues to be of importance in the field of VT ablation. However, some have adopted these findings to the degree that "empiric" ablation of voltage channels is performed as part of a VT ablation procedure,